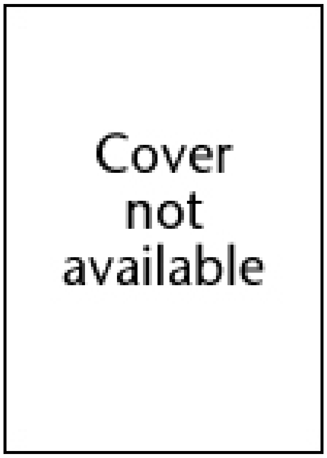

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An Outbreak of Hypersensitivity Pneumonitis at a Metalworking Plant: A Longitudinal Assessment of Intervention Effectiveness

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The authors describe a longitudinal assessment of intervention effectiveness in response to an outbreak of hypersensitivity pneumonitis (HP) at a metalworking facility. Thirty-five (29%) of the plant's 120 production workers were given a clinical diagnosis of HP during the two years of the investigation. Although quantitative exposure assessment tools were of limited utility, the investigators successfully used qualitative observations and the patients' return-to-work experiences to iteratively evaluate their exposure control recommendations. Recommended interventions included improving metalworking fluid management practices, enclosing selected metalworking fluid machining operations, eliminating mist cooling, exhausting two additional water-based industrial processes, increasing general dilution ventilation, and worker training. As of November 1999, 26 months into the outbreak, 51 percent (18) of the employees with a clinical diagnosis of hypersensitivity pneumonitis had been able to return to work. The symptom onset of the 35 workers who were given a clinical diagnosis of hypersensitivity pneumonitis during the two-year study period predated the implementation of the interventions. The collaboration of a multidisciplinary team appears to have allowed for successful intervention in this setting. A specific etiological agent(s) associated with the outbreak was not confirmed during the investigation. An acid fast isolate identified as being in the *Mycobacterium chelonae* group was detected in only one of the submitted metalworking fluid (MWF) sump samples. Longitudinally, there was a statistically significant difference in MWF sump bacteria ($X^2 = 286.4$, df $= 17$, $p < .0001$) and MWF sump fungi ($X^2 = 28.1$, df $= 7$, $p < .0002$). Measured oil mist air levels did not exceed the Occupational Safety and Health Administration's (OSHA's) permissible exposure limit (PEL), and in fact, did not exceed 0.5 mg/m^3.

Keywords Hypersensitivity Pneumonitis, Metalworking Fluids, Intervention Effectiveness

A sentinel case of hypersensitivity pneumonitis (HP) was diagnosed at a university-based occupational and environmental medicine clinic in fall 1997. Thirty-four additional workers from the same factory were given a clinical diagnosis of HP during the subsequent two years. The demographics, symptoms of the workforce, and diagnostic strategies have been presented elsewhere.[1,2]

The small non-union manufacturing facility (67,000 ft^2 production area, 120 production employees) machined parts from titanium and high nickel alloy. After an electrochemical-machining (ECM) step, parts were machined, polished, and inspected in an open production area. Many employees worked for more than 40 hours a week. The index case, a machinist, and the other clinically diagnosed cases worked in this open production area.

Because a sentinel case of occupational illness serves as a warning that exposure controls are inadequate, an industrial hygiene evaluation of the workplace was initiated.[3] In a previous publication, the industrial hygiene results from the first nine months of this outbreak were compared to the industrial hygiene data collected at a control plant.[1] This article explores an iterative approach to exposure control and disease prevention over a two-year period in the facility with the outbreak.

Hypersensitivity pneumonitis (HP) is an interstitial lung disease caused by an immune response to inhaled antigenic particles. Symptoms include cough, chest tightness, dyspnea, chills, fever, malaise, weight loss, and progressive dyspnea. The responsible antigens are usually of bacterial, fungal, or animal protein origin. Small inorganic compounds also can induce HP when they act as haptens and bind to serum protein to create an antigenic complex.[4]

Since 1993, over 12 outbreaks of HP have been reported in metalworking environments.[5–13] In these settings, it was postulated that the outbreaks were associated with airborne (inhalation) exposure to microbially contaminated water-based metalworking fluids (MWF). Although some of the investigators attributed the outbreaks to *Pseudomonas fluorescens*,[10] *Mycobacterium chelonae*,[7,9,11] *Acinetobacter* sp.,[12] and *Mycobacterium immunogenum*,[13] a specific microbial antigen, agent, or set of agents has not been confirmed in metalworking settings.

The researchers who evaluated the patients' work site during this investigation were confronted with the challenge of completing an exposure assessment and making intervention recommendations in a setting with no confirmed etiological agent(s). Although microbially contaminated metalworking fluid was considered the most likely exposure source, other microbially contaminated water-based sources were evaluated as well. Because there were no specific agents to measure, criteria-based exposure assessment had little utility.

The approach taken by the investigators highlights the importance of multidisciplinary collaboration among industrial hygienists, occupational physicians, epidemiologists, the employer, and the employees in the management of occupational disease during a cluster or sentinel health event investigation.[14] This collaboration appears to have allowed for successful intervention in this setting. The implementation of a comprehensive metalworking fluid management program and the control of aerosols from metalworking machines and other water-based "point sources" were important control strategies.

METHODS

Patient Management

Employees with respiratory or systemic symptoms consulted physicians in the University's Occupational and Environmental Medicine Unit. The initial cases ($n = 16$) were diagnosed with HP based on respiratory and systemic symptoms and a lung biopsy consistent with HP. Subsequent HP cases diagnosed without a biopsy were based on a combination of symptoms, physical findings, radiological findings, pulmonary physiology, and response to removal from work.[1]

When a diagnosis of hypersensitivity pneumonitis was made, decisions regarding return to work involved the physician, the industrial hygienist, the patient, and the employer. After reviewing the implementation status of recommended interventions, physicians carefully discussed with patients the likely consequences of return to work, options (if any) of restricted work, and the process involved in seeking workers' compensation benefits if a patient was not safely able to return to work. If patients returned to work they were re-evaluated on a regular basis.

Environmental Assessment

In parallel with the clinical evaluations of the HP cases, the investigators completed a qualitative and quantitative industrial hygiene assessment of the work environment and made intervention recommendations based on their findings and the patients' return-to-work experiences. After the implementation of some of the recommended interventions the investigators continued to collect longitudinal data. The qualitative assessment of exposure involved the identification of potential sources and the documentation of exposure control strategies through observations and checklists. The quantitative assessment of exposure relied on the collection of air and source samples. These exposure assessment strategies are described below in more detail.

Qualitative Exposure Assessment

Over a two-year period (fall 1997 to fall 1999), the investigators conducted multiple walk-through surveys of the plant. The investigators used a checklist survey to evaluate each metalworking machine. The checklist (Appendix A) included questions concerning metalworking fluid management. The investigators modified the checklist from the instruments initially developed by the National Institute for Occupational Safety and Health (NIOSH)[15] and by the Organization Resource Counselors (ORC).[16] Because NIOSH and the ORC emphasize the importance of maintaining metalworking fluids with a comprehensive coolant management plan, the investigators reviewed coolant management practices throughout the study period.

The investigators identified other water-based industrial processes during their walk-through surveys of the plant. They documented the control strategies used to control mist from these processes. In addition, the investigators reviewed the design, operation, and maintenance of the ventilation systems in the factory.

Quantitative Exposure Assessment

General Overview

Traditionally, metalworking fluid exposure has been measured by sampling for mineral oil mist. Although oil mist air levels were documented in this plant, the primary focus of the quantitative exposure assessment strategy was on characterizing microbial contaminants in water-based reservoirs and in the air. The investigators collected bulk samples from fluid reservoirs (machine sumps and other water-based reservoirs). In addition, the investigators collected area bioaerosol samples. Samples were collected before and after the implementation of interventions.

Because of analytical cost considerations (64 separate metalworking sumps used water-based metalworking fluids), representative bulk samples were analyzed for bacteria, fungi, endotoxin, and mycobacteria. The bulk MWF samples' physical characteristics (e.g., pH, tramp oil, and concentration) were recorded when possible. In different zones of the plant, area air samples were collected for viable bacteria, viable fungi, and endotoxin. The zones represented the work locations of the index patients. The sampling and analytical methods are described below.

Bulk Samples

Bulk samples were collected in sterile containers from metalworking sumps and other water-based processes longitudinally during the study period. Bulk sump samples were collected from the metalworking machines of index patients and selected machines representing different coolant types, zones, and machine types. Bulk MWF samples were serially diluted, inoculated onto 2 percent malt extract agar (MEA), for fungi, tryptic soy agar (TSA), bacteria, and either Middlebrook 7H11 agar or "myco agar," for mycobacteria. All plates were incubated at 25°C.

For aerobic bacteria and fungi, plates were checked in 5 to 7 days. Selected colonies were then sub-cultured and purified for identification and speciation. For mycobacteria, plates were checked twice a week. Negative results were declared only after at least one month of observation. Some bulk samples were stained and viewed microscopically before culture. The employer collected additional bulk samples. Some of these samples were semi-quantitatively analyzed for microbial contamination with commercial hygiene contact slides (Difco Hycheck, Detroit, MI). The plate count agar slides were incubated at 35°C–37°C.

Bulk samples representing different coolant types, zones, viable bacteria concentrations, and machine types were submitted for endotoxin analysis. These samples were assayed for endotoxin content using a Limulus amebocyte lysate (LAL) assay (Kinetic-QCL, Biowhittaker Inc., Walkerville, MD). Aggregate bulk samples were cultured for thermophilic bacteria. These samples were plated on TSA at 55°C.

Air Samples

At the onset of the investigation and after each major intervention, microbial samples were collected in several production areas of the plant and outdoors. The sampling locations represented the work locations of the index patients. An Andersen N-6 sampler calibrated to 28.3 liters per minute (Lpm) was used to collect the samples. Paired samples were collected from 30 seconds to 2 minutes on either MEA or TSA.

Bioaerosol samples were collected when water-based metalworking machines and other water-based industrial processes were not in operation. These samples represented background or "quiescent" bioaerosol concentrations. Sampling was repeated in the same locations during the normal operation of these water-based machines and processes. These samples represented "semi-aggressive" bioaerosol concentrations. A comparison between "quiescent" and "semi aggressive" bioaerosol concentrations allowed for a longitudinal assessment of the impact of the operation of potential sources on bioaerosol concentrations.[17,18] The samples were cultured for viable bacteria and fungi on TSA and MEA at 25°C.

A Miniram Personal Monitor Model PDM-3 was used to characterize area aerosol concentrations in different zones of the plant during quiescent and semi-aggressive conditions. The Miniram was factory-calibrated to sample particulate in the respirable and inhalable size fraction, 0.1 to 10 micrometers (μm) in diameter.

Airborne endotoxin levels were measured on three different dates. The samples were collected on polycarbonate 0.40 μm capillary-pore membranes in three-piece polystyrene cassettes with glass fiber backup pads. The samples were collected for 4–5.5 hours at 2.3 Lpm. The samples were analyzed using the kinetic limulus assay with resistant-parallel-line estimation (KLARE) method.[19] On three additional consecutive dates, airborne endotoxin levels were measured using PVC filters and teflon-coated glass fiber filters. The air samples were collected for 6–8 hours at 1 cfm. The filters were aseptically transferred to sterile 50 milliliter centrifuge vials and extracted using 10 ml of pyrogen-free water. The samples were then centrifuged and the supernatant fluid was recovered and stored at −85°C until it was assayed for endotoxin content. Different endotoxin sampling methods were used during the two-year study period because one lab was involved in the initial assessment phase and a second lab was involved during the longitudinal assessment phase.

Four area oil mist air samples were collected in the grinding department, the region of the plant that had been identified with the Miniram as having the highest aerosol concentrations. Oil mist samples were collected on preweighed polyvinyl chloride (PVC) filters at 3 Lpm for four hours. The samples were analyzed using gravimetric (NIOSH Method #0500) and infrared spectroscopic analyses (NIOSH Method #5026).

Statistical Analysis

Data were analyzed using SPSS for Windows. Standard statistical parametric (bivariate correlations, t-test, analysis of variance) and non-parametric (chi-square [χ^2], Mann-Whitney U [MWU], and Kruskal-Wallis [KW]) univariate tests were used. Data were plotted and transformed logarithmically, where appropriate. Significance levels were set at $p < .05$.

RESULTS

The study results are presented in four sections. Initial qualitative and quantitative exposure assessment data are summarized in the first section. The recommended interventions that were proposed after the initial exposure assessment are reviewed in the second section. The third section presents the qualitative and quantitative exposure assessment data that were collected after the implementation of interventions. The clinical course of affected workers is summarized in the fourth section.

Initial Exposure Assessment

Initial Qualitative Assessment

Machining. The checklist survey provided the investigators with information about the plant's coolant management program. At the beginning of the study period, 64 of the 300 metalworking machines used water-miscible metalworking fluids. These manual and automatic machines (lathes, milling machines, sawing machines, grinders) used two soluble, one semisynthetic, and one synthetic MWF. The soluble and semisynthetic MWFs were pumped from separate 20–250 gallon reservoirs through nozzles that flooded the tools, the workpieces, and the chips. The majority of the machines had sumps of 50 gallons or less. The synthetic MWF was sprayed onto parts as a mist. The soluble and semisynthetic MWF concentrates contained biocides; the synthetic did not. There had not been a major change in the production process that could be temporally associated with the onset of the HP outbreak.

Employees' responses to the checklist survey suggested that the company's MWF coolant management program was decentralized. Most machinists made their own coolant additions as coolant was depleted from their machines. Although machinists added defoamers to the sumps, they did not add biocides. Some machinists used a refractometer to maintain the correct coolant concentration. Circulating coolants were filtered with mesh screens and, in one case, a cloth filter. Although oil skimmers were not available, machine maintenance successfully controlled the concentration of tramp oil in some machines. The company's drain, clean, and recharge program did not include a rigorous cleaning protocol with sump and circulation system flushing. In addition, more than half of the machining operations were not controlled with mist collection.

Other water-based industrial processes. Two additional water-based industrial processes were identified as potential source risks during the walk-through surveys. Three months into the outbreak, the investigators identified a 20,000 cfm wet dust collector as a potential reservoir for microbial contamination. Six months later they identified a water-based abrasive blasting process (vapor blasting) as an additional microbial source. Emissions from the wet dust collector and the vapor blast were not exhausted outside.

Ventilation. During the fall and spring, dilution ventilation was supplied to the production area of the plant through 11 rooftop heating ventilating and air-conditioning (HVAC) units. During the heating and cooling seasons (winter and summer) there was no dilution ventilation because the HVAC's outside air louvers were closed and outside air was not mixed with returned air. The HVAC's cooling system had been brought back into service during the summer of 1997. The operation of the heating or cooling system did not appear to be temporally associated with the resolution or exacerbation of employees' symptoms.

Initial Quantitative Exposure Assessment

Bulk samples. Results of initial bulk sampling are presented in Table I. No clear "point source" or problem sump was apparent. Based on initial sump sampling, sump bacteria concentrations were not associated with machine type, date of MWF replacement, pH, MWF type, or the zone where the machine was located. Worker perception of sump tramp oil contamination was significantly associated with sump bacterial concentrations ($n = 19$; $r = .467$; $p = .044$). For seven paired samples, sump bacteria concentrations measured with dipsticks were correlated with

TABLE I
Initial quantitative exposure data

	Bulk samples—machining sumps	Air samples—indoors	Air samples—outdoors
Bacteria	Mean: 5.4 10^7cfu/ml Range:10^5–10^8 cfu/ml (LOD = 110 cfu/ml) n =19	Mean: 930 cfu/m^3 Range: 354–2,048 cfu/m^3 (MDC = 18 cfu/m^3) n = 3 paired samples	Mean: 120 cfu/m^3 Range: <53–230 cfu/m^3 (MDC = 53 cfu/m^3) n = 3 paired samples
Fungi	Mean: 376 cfu/ml Range: ND–2970 cfu/ml (LOD = 110 cfu/ml) n = 19 Taxa rank: *Geotrichum candidum* *Fusarium* *Acremonium*	Mean: 394 cfu/m^3 Range: 265–530 cfu/m^3 (MDC = 18 CFU/m^3) n = 3 paired samples Taxa rank: *Basidiomycetes* *Penicillium* *Cladosporium*	Mean: 1412.5 cfu/m^3 Range: 847.5–1854 cfu/m^3 (MDC = 18 CFU/m^3) n = 3 paired samples Taxa rank: *Basidiomycetes* *Cladosporium* *Penicillium* *Epicoccum nigrum*
Endotoxin	Mean: 53,758 EU/ml Range: 7.2×10^3–2.0×10^5 EU/ml (LOD = 0.05 EU/ml) n = 9	Mean: 16.625 EU/m^3 TWA Range: 1.3–58.1 EU/m^3 (MDC = 0.05 EU/m^3) n = 4	<0.06 EU/m^3 TWA (MDC = 0.05 EU/m^3) n = 1
Oil mist		Mean: 0.09 mg/m^3 TWA Range: 0.05–0.18 mg/m^3 (MDC = 0.02 mg/m^3) n = 4	

the bacteria measurements reported by the analytical laboratory ($r = .802$, $p = 0.03$).

Viable bacteria grew in water and sludge from the wet dust collector and the vapor blast. Concentrations in these reservoirs ranged from 10^6 to 10^8 cfu/ml (limit of detection [LOD] = 110 cfu/ml). The bacteria species identified in bulk samples from MWF, the wet dust collector and the vapor blast with the methods described above are summarized in Table II. Similar microbial species were identified in each of the three bulk sources. Similar to what has been described in other investigations,[20–22] several species of pseudomonas were identified in the MWF sumps.

Fungi were cultured from MWF bulk samples (Table I). In addition to the MWF sumps, fungi were cultured from wet dust collector sludge (7×10^3 cfu/g) and the vapor blast (220 cfu/ml).

In April 1998 (7 months into the outbreak) an acid fast isolate from a milling machine's misting fluid was identified as being in the *Mycobacterium chelonae* group. This centrally located machine used a synthetic MWF. The MWF bulk sample in which *M. chelonae* was detected had a mycobacteria agar count of 10^3 cfu/ml and a viable bacteria concentration of 10^6 cfu/ml. Viable *M. chelonae* were not detected in any of the other submitted metalworking sump samples to an LOD of 1 cfu/ml.

TABLE II

Summary of bacteria species identified in MWF, wet dust collector, and vapor blast bulk samples

Dominant bacteria species	MWF sumps	Wet dust collector water	Wet dust collector sludge	Vapor blast slurry
Acinetobacter baumannii		√		
Acinetobacter calcoacetius				√
Acinetobacter lwoffii	√	√		
Aeromonas hydrophilia group		√	√	
Alcaligenes piechaudii	√			
Alcaligenes sp.	√		√	√
Bacillus sp.*	√	√		
Brevundimonas diminute		√		
Brevundimonas vesicularis		√		√
Burkholderia capacia			√	
Cedecea davisae	√			
Chromobacterium violaceum	√			
Citrobacter diversus	√			
Comamonas acidovorans				√
Comamonas testosteroni	√			
Coryneform bacteria		√		√
Enterobacter amnigenus 1	√			
Enterobacter gergoviae	√			
Flavobacterium breve	√	√		
Flavobacterium sp.	√	√		
Micrococcus sp.*	√			
Moraxella nonliquefaciens		√		
Moraxella osloensis	√	√		
Moraxella sp.	√	√		√
Oligellaurethralis	√			
Pasteurella-Actinobacillus sp. SF	√			
Pseudomonas aeruginosa	√			
Pseudomonas pseudoalcaligenes	√	√	√	
Pseudomonas putida	√			
Pseudomonas stutzeri	√			
Pseudomonas cepacia	√			
Pseudomonas pickettii	√			
Shewanella putrefaciens	√	√		
*Staphylococcus epidermidis**	√			
*Streptococcus salivarius**	√			
Vibrio sp. SF		√		

*Gram-positive bacteria.

M. chelonae were not detected in vapor blast samples. Water from the wet dust collector and four MWF bulk samples had evidence of acid fast bacteria on stained slides but no culturable mycobacteria.

Air sampling. Initial air sampling data are summarized in Table I. Airborne bacteria and endotoxin concentrations were greater indoors than outdoors. Airborne fungi concentrations were lower indoors than outdoors. The taxa rank of fungi detected indoors was similar to the taxa rank of the fungi detected outdoors. Measured oil mist levels were consistently below OSHA's Permissible Exposure Limit (PEL) (5 mg/m^3).

All three water-based point sources produced an aerosol measurable with the Miniram (MDC = 0.01 mg/m^3). The highest aerosol levels, 0.8 mg/m^3 were measured in the machining area with the most grinding operations. The operation of metalworking machines, the water-based dust collector, and the vapor blast was associated with an increase in bacterial levels (nearly 12-fold) and an increase in particulate levels (3.7-fold). When these reservoirs were operated there was no difference in airborne fungal levels.[1]

Several models were developed to explore the relationship between the presence of disease and various exposure surrogates: sump bacterial concentration, work zone, job title, mist control, and a variety of calculated indices. No associations were identified.

Recommended Interventions

Recommended interventions were designed to meet two complementary goals. One goal was to remove the suspected yet unconfirmed microbial contaminant(s) from metalworking fluid reservoirs. The second goal was to reduce the total concentration of bioaerosols in the plant. As a result, proposed interventions included the implementation of a comprehensive metalworking fluid management program, machine enclosure with either outside exhaust or recirculation through appropriate mist collectors, and an increase in general dilution ventilation. Administrative controls were recommended as well. These recommended controls included worker training and a reduction in the number of overtime hours worked.

The investigators proposed additional interventions in response to initially unsuccessful employee return-to-work initiatives (discussed below). These intervention proposals included the recommendation that emission from the other water-based reservoirs, the wet dust collector, and the vapor blast, be exhausted out of the plant.

Exposure Assessment after the Implementation of Interventions

Qualitative Assessment: Post-Intervention

As discussed below, the employer implemented many of the recommended interventions. In addition, they introduced additional exposure control strategies of their own.

Machining. The employer drained and recharged all of their MWF sumps in December 1997 and implemented a comprehensive coolant management program in April 1998 (7 months into the outbreak). All MWF machines were repeatedly drained, mechanically cleaned, disinfected with machine cleaner, and recharged with new coolant through a centralized MWF mixing and management program. Through their new coolant monitoring initiative (pH, concentration, bacteria) the employer evaluated the efficacy of a monoethanolamine pH adjuster (introduced in April 1998), a new synthetic coolant (introduced in some machines in November 1998, 14 months into the outbreak), and the effectiveness of an oil skimmer.

Because an acid fast isolate from the *M. chelonae* group was identified in a milling machine that used a mister, the employer eliminated mist cooling in May 1998 (8 months into the outbreak). The synthetic coolant used in these machines was discontinued and replaced with a soluble oil coolant with a flood delivery system. Where straight oil use was possible, the company completely eliminated the use of water miscible coolants. By April 1998 the total number of machines with water-based coolant had dropped from 64 to 52. By May 1998 the number had dropped to 34.

In April 1998 engineering controls were introduced. The company enclosed its eight grinding machines with shrouds and exhausted their emissions (3200 cfm) out of the plant. The grinding machines were targeted for mist and dust control because they generated the most aerosol. The elevated generation of mist in grinding operations is consistent with other machining settings.[22] Mist control was not implemented on the remaining metalworking machines.

Other water-based industrial operations. The wet dust collector and the vapor blast were exhausted out of the plant in April 1998 (7 months into the outbreak) and August 1998 (11 months into the outbreak), respectively.

A separate positive-pressure room with its own air-handling unit was identified for the polishing operations that used the wet dust collector. By May 1998 employees polishing titanium were transferred to this room. Although some polishers elected to wear disposable dust masks, a comprehensive respiratory protection program with fitted facepieces was not introduced in the plant.

Plant-wide administrative controls. Some employees were able to reduce the number of overtime hours worked. Others continued to work overtime. Throughout the investigation, plant management and the staff from the occupational and environmental medicine unit held training sessions, or "town meetings." At these meetings the general health of the workers and the status of implementing controls were discussed.

Ventilation. After the outside exhaust of the wet dust collector, the grinding machines, and the vapor blast, the plant operated under negative pressure. The outside air dampers for the roof top units were manually set to be open a minimum of 20 percent at all times.

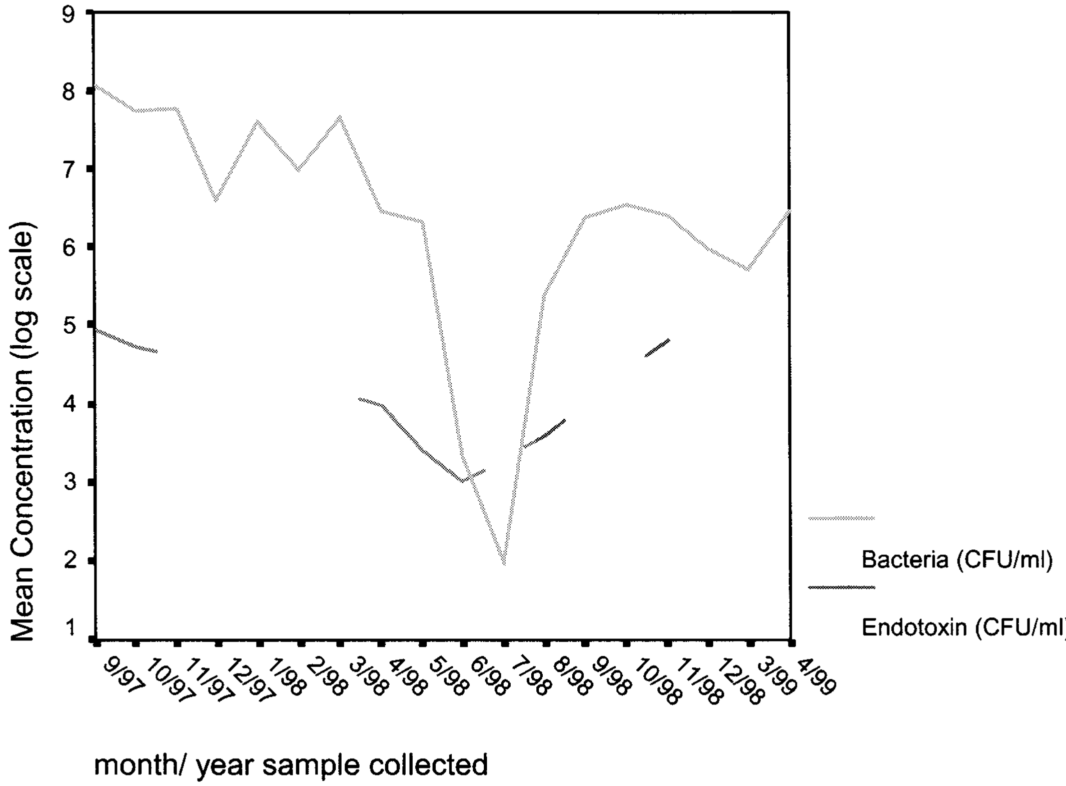

FIGURE 1

Machining sumps: log of mean bacteria and log of mean endotoxin, September 1997–April 1999.

Quantitative Assessment: Post-Intervention

Bulk samples. A statistically significant difference was found in MWF sump bacteria between October 1997 and April 1999 ($X^2 = 286.4$, df $= 17$, $p < .0001$) (Figure 1). During this time period there was not a statistically significant difference in sump endotoxin levels. Over the study period, as reported in other settings, sump bacteria concentrations, and sump endotoxin concentrations were correlated ($r = 0.609$, $p < .01$).[(23)] There was a dramatic decrease in sump bacteria concentration between April 1998 and June 1998. Although the log of the mean concentration of sump bacteria increased between July 1998 and October 1998, the log of the mean concentration remained at least two orders of magnitude below the log of the initial mean sump bacteria concentration.

Between October 1997 and November 1998 there was a statistically significant difference in MWF sump fungi over time ($X^2 = 28.1$, df $= 7$, $p < .0002$). By November 1998 the mean concentration of fungi in sumps had declined to 135.5 cfu/ml (SD $= 365$). After January 1998, fungi were rarely detected in the MWF sumps.

In November 1998 (14 months into the outbreak) and November 1999 (26 months into the outbreak), *Mycobacterium gordonae* was detected in bulk samples of water from the water-based dust collector and vapor blast slurry. Acid fast bacilli from the *M. chelonae* group were not detected in any of the submitted post-intervention MWF, dust collector, or vapor blast bulk samples. In November 1999, with the exception of the wet dust collector sludge, thermophilic bacteria were not detected in bulk samples.

Air samples. Over the study period there was not a statistically significant change in the concentration of airborne fungi or bacteria measured during "semi-aggressive" conditions. Nor was there a statistically significant change in the absolute or percent difference in airborne bacteria or fungi over time when air levels measured during quiescent conditions were compared to air levels measured during semi-aggressive conditions. There was no relationship between the concentration of culturable bacteria in point sources (i.e., reservoirs) and either the absolute or percent increase in airborne bacteria levels when air levels measured during quiescent conditions were compared to air levels measured during semi-aggressive conditions.[(17,18)] Data from selected zones are summarized in Table III.

After the introduction of interventions, aerosol concentrations measured in production areas with the Miniram ranged from 0.01–0.235 mg/m^3 (MDC $= 0.01$ mg/m^3). Over time there was a statistically significant decline in aerosol levels measured during semi-aggressive conditions ($X^2 = 11.9$, df $=$ 4, $p < .018$). However, when samples measured during semi-aggressive conditions were compared to samples measured during quiescent conditions, there was not a statistically significant association between the proportional increase in airborne aerosol levels and the proportional increase of airborne bacterial levels.

TABLE III
Mean source strength (CFU/ml) and absolute/percent increase in bacteria air levels

	Bacteria														
	October 1997[B]			April 1998[C]			May 1998[C]			June 1998[C]			November 1998[C]		
Zone[A]	Mean source strength (SD)	Absolute increase	Percent increase	Mean source strength (SD)	Absolute increase	Percent increase	Mean source strength (SD)	Absolute increase	Percent increase	Mean source strength (SD)	Absolute increase	Percent increase	Mean source strength (SD)	Absolute increase	Percent increase
Zone 3	5.6 10^7 (3 10^7)	282.5	16	55 (0.000)	1051	1480.28	55 (--)	5777.5	5450.47	55 (--)	53.25	300	3 10^6 (3 10^6)	−988.5	−95.6
Zone 4	2 10^7 (4760710)	159.5	82.22	302.5 (350)	26.50	13.05	55 (--)	344.5	782.95	55 (--)	110	275	2 10^4 (8 10^3)	92.7	697
Zone 7	4.4 10^7 (5.5 10^7) MWF			1.2 10^7 (1.4 10^7) MWF + WDC	3507	1072.48	16142.5 (22751) MWF + WDC	10433	6228.66	5021770 (7101714) MWF + VB	−105.5	−47.85	1938.5 (4919.4) MWF + WDC + VB	5765	50.3

[A]Zones:
Zone 3-production/ central plant. Sources (reservoirs) = MWF sumps.
Zone 4-production/ central plant. Sources (reservoirs) = MWF sumps.
Zone 7-production/ near vapor blast and room with wet dust collector. Sources (reservoirs) = MWF sumps, Wet Dust Collector (WDC), and Vapor Blast (VB).
[B]Samples collected after one machine had not been operating for at least one hour and again after ten minutes of operation.
[C]Samples collected at night with all machines turned off and again during the day after several hours of machining.

After the introduction of interventions, airborne endotoxin concentrations in production areas of the plant ranged from 0.98–2.92 EU/m^3. There was a higher level of airborne endotoxin in production areas versus non-production areas (Mann-Whitney U comparison = 5.0, p = .004). The airborne endotoxin concentration in a non-machining production area was not statistically different than the airborne endotoxin concentration in machining production areas. Similarly, the airborne levels of bacteria, fungi, and particulate measured during semi-aggressive conditions in non-machining production areas were not statistically different than their airborne levels measured in machining production areas.

Clinical Experience of HP Cases

Workers who were diagnosed with hypersensitivity pneumonitis were removed from the plant with their concurrence. Those who recovered over weeks to months were initially offered a return to work as the team documented that environmental conditions improved. In January 1998 (4 months into the outbreak) several workers returned with prompt recurrence of their symptoms and a decline in their lung function. As discussed above, additional interventions were implemented and in June 1998 (9 months into the outbreak) another attempt at return to work was successful for some but not for others. For the group that unsuccessfully returned to work in June 1998 the clinical response was more subtle. Further interventions were implemented and after August 1998 more individuals were able to return to work. As of November 1999, 26 months into the outbreak, 51 percent of the employees with a clinical diagnosis of hypersensitivity pneumonitis had successfully returned to work. A detailed discussion of the employees' return to work experience will appear elsewhere.

A cross-sectional survey completed in November 1998 identified two possible HP cases who did not attend the clinic and who reported symptom onset in July and August of 1998.[(24)] The symptom onset of the HP cases recognized during these two years of the outbreak investigation predated the implementation of the April/May 1998 interventions.

DISCUSSION

After a cluster of hypersensitivity pneumonitis cases was recognized at an occupational medicine clinic, the investigators initiated an industrial hygiene investigation at the patients' metalworking facility. The majority of the proposed interventions were designed to reduce employees' inhalation exposure to the bioaerosols associated with machining with water-based MWF and the operation of the vapor blast and wet dust collector. Proposed interventions were focused on improvement in coolant management practices and the reduction of aerosol emission from identified water-based point sources. Because the interventions were implemented concurrently, it was not possible to evaluate quantitatively the effectiveness of each intervention in isolation.

The industrial hygiene evaluation was done in parallel with the clinical evaluation/medical management of the company's employees. Patients served as the primary source of information justifying return to work or continued removal, complementing the measurement of exposures and the development of additional intervention strategies. Communication was an essential part of this iterative model of intervention. Prompt feedback when symptoms worsened led to searches for objective (physiological) data to confirm the suspicion of worsening disease.

In the absence of such supporting evidence, patients returned to the workplace under three conditions. First, they understood that although individual tests might provide false negative results, they would be re-evaluated on a regular basis. Second, their healthcare providers served as their advocates in discussions with the insurers and employer. Third, they understood that the team managing the outbreak had the full confidence of plant management. Town meetings reduced fear and encouraged participation in the clinical evaluation, even when this led ultimately to job loss. This communication also allowed some workers to retain jobs, as they could be reliably assessed over time. Although "advocacy" for workers has often been perceived as fractious, in an outbreak of this magnitude only parallel clear statements of advocacy for health and fundamental adherence to scientific principles, together with modern risk communication approaches, provided ill employees the certainty that their clinical needs would not be superseded by economic or political decisions without their knowledge or influence.

As of November 1999, 51 percent of the employees with a clinical diagnosis of HP had been able to return to work. The clinical response of the HP cases that returned to work was progressively more subtle as additional interventions were implemented. The symptom onset of all of the clinical HP cases recognized during these two years of the outbreak investigation predated the implementation of the April/May 1998 interventions. This clinical experience may point to the effectiveness of the implemented interventions for a percentage of the plant's workforce. Conversely, the responsible etiological agent(s) may have declined or disappeared for reasons unrelated to the control strategies described in this article.

The quantitative evaluation of the effectiveness of the proposed interventions was challenging because a specifical etiological agent(s) associated with the outbreak was not confirmed during the study period.[(25)] However, as discussed in a classic epidemiology text and a recent article,[(26–27)] the history of public health has shown that industrial hygienists do not need to understand causal mechanisms completely to introduce prevention measures. In the absence of quantitative data, a qualitative assessment of potential exposures has value.

Improvement in sump management was documented through the qualitative review of sump management practices. The checklist survey was a useful evaluation tool. Coolant management checklists have helped other employers improve their MWF management programs. The statistically significant difference in viable sump bacteria and fungi indicated a change in sump management practices as well. The most dramatic decline in measurable sump bacteria was recorded after the employer had first introduced a comprehensive sump management program that included the use of a pH adjuster. Although there was a statistically significant difference in sump bacteria over the study period, the increase in bacteria after the initial dramatic decline may have been because the concentration of the pH adjuster was too low or its use permitted the growth of other species of bacteria.[28]

Although a decline in viable sump bacteria from 10^8 to 10^6 is consistent with the goals outlined in the ORC's original guidance document,[16] the adoption of a universally recommended bacteria concentration in MWF remains controversial. From a health perspective, the species of bacteria that grow in metalworking sumps may be as important as the total concentration of viable bacteria. As a result, the routine monitoring of sumps without bacteria speciation may have limited utility. In addition, the random use of biocides to keep bacteria below an arbitrary concentration should be discouraged. Although biocide use may be appropriate under certain circumstances, it is important to note that biocides may kill off one species of bacteria and lead to the preferential growth of a species with more pathogenic properties.[29] The concentration and type of bacteria species found in the MWF sumps of small machine shops remain important research questions.

The machine operators' perception of tramp oil was the only variable that was significantly associated with the concentration of gram-negative bacteria in the sumps. Sump management practices that include tramp oil reduction through machine maintenance and oil skimming are important control strategies.[30] The association between tramp oil reduction and microbial levels has been cited in other studies.[31] OSHA's Standard Advisory Committee (SAC) has proposed a comprehensive MWF standard that includes a systemic approach to sump management.[32] OSHA's MWF Best Practices manual recommends this approach as well.[33] The investigators' experience with managing the response to this outbreak underscores the importance of a systems approach to exposure assessment and control.

Similar microbial species were identified in the MWF sumps, the wet dust collector, and the vapor blast. Microbial contaminants from one of the water reservoirs may have become entrained into one of the other water reservoirs, increasing the potential for microbial amplification and distribution. Consequently, the control of emission from all water-based point sources appears to have been essential.

An acid fast isolate from one of the milling machines was identified as being in the *M. chelonae* group. This milling machine applied a synthetic coolant with a mister. Although viable *M. chelonae* was not identified in any of the other submitted samples, it may have grown in one or more of the sumps from which a sample was not collected. Alternatively, the analytical methods used by some of the labs may not have had the specificity to detect this organism. The presence of an acid fast isolate from the *M. chelonae* group may have been significant because *M. chelonae* has been identified as a suspected etiological agent after other outbreak investigations.[9,11] Additionally, the acid fast isolate identified in this investigation may belong to a new taxon, *M. immunogenum*, that has been proposed after the genetic analysis of isolates from other outbreaks.[13,34,35]

The implementation of selected mist control strategies was documented with qualitative exposure assessment tools. Although bioaerosol exposure data were collected before and after interventions had been initiated, the investigators were not able to document a statistically significant reduction in airborne viable bacteria or fungi exposure levels during semi-aggressive conditions. Given the variability in machining, wet dust collection, and vapor blast conditions, the longitudinal collection of bioaerosol data had little utility. However, the initial bioaerosol samples did document an association between airborne bacteria and the operation of metalworking machines, the wet dust collector, and the vapor blast. This documentation reinforced the recommendation to control aerosol emission from all water-based point sources.

The lack of an association between the absolute or percent difference of airborne bacteria during quiescent and semi-aggressive sampling conditions and the source strength of bacteria in identified point sources raises important questions about the process of aerosolization and bioaerosol sampling strategies. In addition to the reservoirs' bacteria or fungi concentration, the measured concentration of airborne bacteria or fungi may be related to additional factors. These factors may include the aerosol generation characteristics of a specific machining process; the design of the machines' enclosures; the distance of the bioaerosol sampler from the machines; the numbers of machines operating at a given time; the bioaerosol contribution of neighboring water-based industrial processes and the room's temperature and relative humidity.[29,31] Given the field nature of this investigation, it was not possible to control for all of these variables. Because a specific etiological agent(s) remains unidentified for hypersensitivity pneumonitis, quiescent/semi aggressive bioaerosol sampling in the setting of an outbreak investigation is of questionable practical utility. In fact, without further discussion of the explicit hypotheses in such sampling, these authors would not recommend dynamic sampling comparisons.

Airborne particulate levels increased with the operation of metalworking machines, the wet dust collector, and the vapor blast. The variability between background/machining ratios measured with the Miniram and similar measurements characterized with microbial sampling may be due to differences in particle size distribution.[36] During this investigation the Miniram was used to measure aerosol levels in different zones of the plant when all machines were either operating or shut off. In future investigations, this approach could be used to target specific machines and operations that generate the most total aerosol.[37]

Compared to data reported elsewhere, airborne endotoxin concentrations were consistently low.[23,38] However, the air levels of endotoxin were higher in production areas than non-production areas. Airborne endotoxin, bacteria, fungi, and particulate levels did not differ in machining versus non-machining zones of the production areas of the plant. These results suggest plant-wide air mixing and offer an explanation for why there was not an association between symptomatic workers and work in any given zone of the plant.

Although the HVAC system represented a potential source for bioaerosol amplification and dissemination, it was not considered the most important source associated with this outbreak. Assessment of HVAC systems during outbreak investigations remains important, however.

The potential synergistic health effects between bioaerosols and other air contaminants in the plant should be explored. For example, polishing operations generate low levels of particulate, metal dust, fibers, and, theoretically, the volatile organic compounds associated with the decomposition products of the wheels.

An additional goal of the industrial hygiene evaluation was to help microbiologists and immunologists define the agents to which employees might be responsive. Immunological data will be presented in a separate publication.[24]

Measured oil mist levels were consistently below OSHA's PEL (5 mg/m^3), NIOSH's REL (0.4 mg/m^3 thoracic particulate mass), and the PEL recommended by OSHA's MWF Standard Advisory Committee (0.4 mg/m^3 thoracic particulate mass).[15,32,33] Compliance with OSHA's oil mist PEL would not have prevented this outbreak. The industrial hygiene approach taken during this investigation underscores the importance of controlling exposures not currently regulated by OSHA. Rather than rely exclusively on criteria-based exposure assessment, a systems approach to exposure controls has merit. This approach has been proposed by OSHA's Metalworking Standard Advisory Committee[32] and has been promoted in OSHA's Metalworking Fluids Best Practices Manual.[33]

CONCLUSION

During this outbreak investigation the most useful exposure information was gathered during the qualitative industrial hygiene assessment of the facility. These qualitative workplace observations reinforced the recommendations to improve coolant management practices, exhaust all aerosol-generating water-based industrial processes, and increase the volume of dilution ventilation. However, the decision to eliminate machining with misters was inspired by the detection of an acid fast isolate from the *M. chelonae* group through quantitative bulk sampling. The majority of recommended interventions were implemented by May 1998, eight months into the outbreak. Because the interventions were implemented concurrently, it was not possible to evaluate quantitatively the effectiveness of each intervention in isolation.

Concurrent with the collection of industrial hygiene data, physicians managed the medical care and return-to-work experience of the sick workers. As of November 1999, 51 percent of the employees diagnosed with hypersensitivity pneumonitis had successfully returned to work. The symptom onset of the clinical HP cases recognized during the two-year study period predated the implementation of the majority of the interventions described here. This clinical experience may point to the effectiveness of the interventions for a percentage of the plant's affected workforce and for the prevention of new cases in the unaffected workforce. The collaboration between industrial hygienists, occupational physicians, the employer, and the employees, allowed for an iterative approach to exposure assessment, the implementation of control strategies, and patient management.

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APPENDIX A: METALWORKING MACHINE SURVEY—OPERATOR CHECKLIST* DATE: _______

Machine Brass Tag Number _______
Machine Type: Grinder _______ Lathe _______ Milling _______ Broach _______ Other _______
Machine Type: NC (numerically controlled) _______ CNC (Computer Numerically Controlled) _______ Manual _______
Metal(s) Machined: ____________
MWF Type: Product Name ____________
MWF Type: Product Category: Straight _______ Soluble _______ Synthetic _______ Semi-synthetic _______
Is anything added to the MWF? Yes _______ No _______
If yes, name of additive(s) ______________________________
Is machine completely enclosed? Yes _______ No _______
If no, does machine have splash guards? Yes _______ No _______
How is the MWF introduced? Flood Coolant _______ Pressurized Coolant _______ Other _______
Sump location: Side _______ Beneath _______
Local Exhaust Ventilation? Yes _______ No _______
If yes, describe: ____________
MWF filtration? Yes _______ No _______
If yes, describe: ____________
Do you monitor the MWFs in any way? (pH? refractometer? Biosticks?) Yes _______ No _______
If yes, describe: ______________________________
Frequency of adding additional MWF ______________________________
Frequency of completely replacing MWF ______________________________
Why is MWF replaced? ______________________________
Who replaces MWF? Machine operator _______ Maintenance department _______ Other _______
Is sump flushed out before MWFs are replaced? Yes _______ No _______
Is circulation system flushed out before MWFs are replaced? Yes _______ No _______
Does tramp oil contaminate the MWF? Yes _______ No _______
If yes, describe: ______________________________
Is machine shut down when not in use? Yes _______ No _______
Does the MWF smell? Yes _______ No _______
If yes, when? ____________
Other observations: ______________________________

*Adapted from the ORC's *Metal Removal Fluids: A Guide to Their Management and Control* (1997).[16]